# Nerve Inflammation Dynamics Underpin Virus Transmissibility and Plegm Migration Required for Onset of Viral Pneumonia Symptoms

17 January 2026
Simon Edwards
Research Acceleration Initiative

## Introduction

Some virii are transmitted through sneezing, including certain virii which cause pneumonia in addition to rhinovirus. Sneezing is most often triggered through the inflammation olfactory nerves, which can be caused by anything from pollen to the effects of a virus. This author has identified two undocumented dynamics which are ultimately related to a virus's ability to either partially or completely prevent nerves from functioning correctly in the nasal cavity.

## Abstract

Although it is already well-understood that nerve inflammation is responsible for sneezing and that this helps certain virii to spread, that inflammation, when it reaches a certain point, actually prevents sneezing. Extreme inflammation of olfactory nerves leads to the loss of the sense of smell, which is also presumably an a priori fact in the research community. Thus, slight inflammation leads to sneezing, but greater inflammation leads to a lack of sneezing and perhaps an impaired sense of smell. We could prove this hypothesis by taking someone with a known airborne allergy and infecting them with viral pneumonia and then seeing if they are able to react to the known allergen at the height of the pneumonia by sneezing.

This extreme inflammation has another after-effect which is not properly understood by the research community. If the nerves in the nasal cavity and pharynx were effectively numbed by the effects of the virus, this might cause the *sensation* of a runny nose to dissipate despite the continued production of a thin mucus which carries the virus in larger quantities. Ordinarily, the sensation of a thin mucus running down the throat would prompt an instinctive swallowing reaction meant to prevent the mucus from entering the airway and causing choking. However, if the nerves were inflammed, this inflammation might indirectly help some small quantities of mucus to enter the trachea and to introduce the virus to that area, leading to bronchitis and, perhaps, even pneumonia.

Not all patients who become partially symptomatic vis-à-vis viral pneumonia develop tracheal involvement and may experience only the sneezing symptom. What this author proposes makes the difference in determining whether a person infected with viral pneumonia develops the more serious symptoms of phlegm in the trachea and water on the lungs is the degree of inflammation in the pharyngeal nerves. If these nerves are only partially impaired, the instinctive swallowing reflex may be activated and this provides some protection against mucus entering the trachea. If they are severely impaired, a patient does not notice the entry of fluid into the trachea and the infection

may, under this condition of numbness, spread into the lower respiratory tract.

The medical community is currently operating under an assumption that viral particles are entering the trachea via aerosol and that mucus actually has a prophylactic effect; trapping viral particles like a filter. Although this is true in one context of a patient who has not yet suffered any degree of infection, once an infection has set in in the pharynx, biological cells from the outer lining of the pharynx detach and are released into the mucus, which is consequently transformed from prophylactic into virus-carrier. I propose that detaching cells from the pharynx deposited into the mucus act to protect viral material from the moisture in the mucus; which would ordinarily be fatal to viral material; in much the same way that bacteria and fungi can be protected by a biofilm. The viral particles are using clumps of dead or detached pharyngeal cells as an impromptu biofilm and the numbing of the pharyngeal nerves to support the successful delivery of the mucus; which acts as a transport system for those affected cells; into the trachea, wherein the virus may be introduced in significant enough quantities to induce the symptoms associated with pneumonia.

Support for this contention lies in the brief return of the sneezing symptom as the coughing symptom abates in those recovering from viral pneumonia.

**Conclusion**

Taking this into consideration, strategies for mitigating the severity of the symptoms of viral pneumonia and for preventing its spread should hinge upon reducing inflammation in the pharyngeal nerves during the early stages of the condition, which are marked by post-nasal drip and sore throat after sleep.